Results and discussion. Injection of 5% glucose (0.2 ml) into the portal vein and into the left carotid artery caused a transient decrease in discharges, whereas the right jugular injection of glucose produced no appreciable change. The effects of glucose were more intense when injected into the cranial side of the carotid artery than into the portal vein. The 0.9% saline infusion produced no response in discharges. These results indicate that the portal glucose infusion effects on efferent activities of the gastric vagus nerve may be attributed to the neural glucose sensitive mechanism located in the portal vein, especially since the same doses of glucose infusion into the right jugular vein had no effect on the discharges (figure, a and b). However, after hepatic vagotomy, the discharge response usually associated with the portal injection of glucose was not reproducible. Regular insulin (4 U i.v.) caused a gradual increase in discharges (figure, c). These experiments were conducted repeatedly, and similar results were obtained. We know that hypoglycemia causes excessive secretion of gastric acid. It has been assumed that hypoglycemia acts by stimulating the neural glucose sensitive mechanism in the central nervous areas, because denervation of the vagus nerve abolishes such response to hypoglycemia[2-5]. However, there are many reports of the existence of a neural glucose sensitive mechanism in the hepato-portal areas. Russek suggested glucoreceptors in the portal areas as a result of his behavioural work[6]. Niijima observed afferent hepatic discharge that was inversely related to the glucose concentration in the portal vein[7]. Schmitt also showed the projection of glucose infusion in the portal vein on hypothalamic neuron activities[8].

In our experiments, glucose infusion into the portal vein, as well as into the cranial side of the left carotid artery, caused a transient decrease in the efferent activities of the gastric vagus verve, whereas i.v. insulin administration caused an increase. These results suggest that the neural glucose sensitive mechanism located in the hepato-portal areas plays a role in these responses, and that there might be a neural system which modulates gastric secretion of acid through hepatic vagal afferents and gastric vagal efferents.

1 Acknowledgment. The authors thank Prof. A. Niijima for his helpful advice.
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Effects of captivity on glucose tolerance in dogs[1,2]

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Summary. Captivity decreased tolerance to glucose and increased blood serotonin levels in 6 normal dogs investigated. Return to freedom brought normalization in the glucose tolerance test and reverted blood serotonin to control levels.

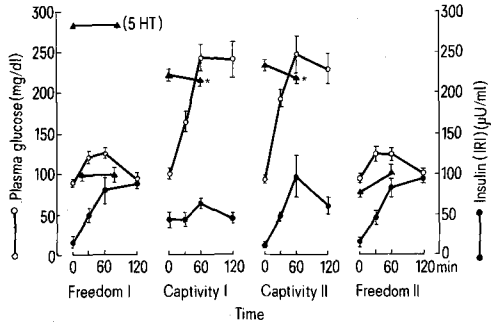
It was found that sulpiride (an atypical dopaminergic blocking agent) but not haloperidol (a classical dopaminergic blocking agent) decreases tolerance to glucose and raises blood serotonin levels both in humans and dogs[4]. Taking into account that these drugs have been successfully used in the treatment of more than 1500 headache and other psychosomatic patients[5,6], and because of some chance findings registered during prior studies, we decided to investigate the effects of captivity on glucose tolerance and on serotonin levels in the blood.

Material and methods. Our protocol included 6 adult mongrel dogs weighing 25–30 kg. Control tests (free state) and captivity state tests were carried out according to plan. When in captivity, animals lived in dog-colony, inside individual barreled boxes measuring $1.25 \times 0.75 \times 0.75$ m. When free (control), animals lived in the garden of our homes, under controlled conditions. During control and captivity periods all animals were fed on a commercial dog food (Perrarina). 4 consecutive (30-day intervals) oral glucose tolerance tests (1.7 g/kg) were performed on each dog. Peripheral blood samples were taken for glucose[7], insulin[8], and serotonin[9] determinations at 0, 30, 60 and 120 min. Whole blood serotonin levels were assessed at 0 and 60 min only. All experiments began at 08.00 h, after 16 h fasting.

Results. Despite the fact that fasting serum glucose levels were normal, captivity reduced the tolerance to glucose, while return to freedom normalized this tolerance (figure). In addition, it was showed clearly that captivity increases blood serotonin levels.

The dogs' behaviour varied according to the experimental conditions. During the first days (3–5) after captivity periods, they were apathetic and did not bark; no responses to environmental stimuli were observed. For instance, they did not react to aggression by other dogs or to children's caresses. However, after those first days, good humour and aggressive manifestations were progressively restored.

Discussion. Captivity decreased tolerance to glucose in the 6 dogs investigated in the present study. Reduction of



4 consecutive oral glucose tolerance test performed in 6 adult mongrel dogs at 30-day intervals. Means±SEM. Serotonin was assayed at 0 and 60 min, only. Changes in 5HT are expressed in percentage. $*p < 0.001$. Statistical significance against zero-mean value at freedom I test (100%).

insulin/glucose ratios, allows us to suppose a diminished response of beta-cells to sugar stimulus. Since exogenous serotonin inhibits glucose-induced insulin secretion[10,11], we think that raised levels of the amine, registered during captivity, could explain the impaired tolerance to glucose.

We found that captivity induced similar effects to those obtained after sulpiride pre-treatment, whereas liberation- and haloperidol-effects were the same[4]. Hence we speculate that low doses of the latter drug provoke catecholamine release from catecholaminergic terminals by blocking presynaptic receptors, which induces hyperactivity of this system[12-14]. On the contrary, sulpiride would block postsynaptic receptors only, inducing dopaminergic system hypoactivity[15,16]. According to the well established antagonism (central and peripheral) between catecholaminergic and serotonergic systems[17], hypo- and hyper-activity of the serotonergic system would correspond, respectively, to the 1st and 2nd of these situations.

The demonstration of dopaminergic[18] and serotonergic[19] fluorescences in beta-cells, in addition to the ability of methysergide maleate (a serotonin antagonist) to enhance insulin secretion[20], reinforces the peripheral antagonism hypothesis, whereas the facts showing that both hypoglycemic and hyperglycemic effects can be induced by brain catecholaminergic mechanisms[21,22] support the central antagonism hypothesis. In addition, it has been shown that the activity of catecholaminergic neurons in the brain and in the peripheral sympathetic nervous system is increased during acute exposure to stress[23]. Stress induces significant depletion of catecholamine stores[24] and predominance of serotonergic neurons[25].

In 2 out of the 6 dogs investigated, captivity was prolonged. Normalization of glucose tolerance tests was registered in 2 experiments performed 3 months after their freedom II tests. Serotonin levels were found normal, also. The fact that animals chronically exposed to stress become resistant to the catecholaminergic depletion[26], is in line with these latter findings.

The 'diabetogenic effect' induced by captivity stress is a complex phenomenon in which other hormones (GH, ACTH, cortisol, catecholamines, glucagon, etc.) and factors like physical inactivity should be considered; however, our results strongly suggest that serotonin plays a role in producing that effect. Since serum prolactin levels were found raised following restraint stress in the rat[27], this hormone should be investigated, also.

1 Acknowledgment. The authors are grateful to Dr P.J. Randle, Dept. of Biochemistry, School of Medicine, Oxford University, for his helpful advices and for revision of the manuscript.
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Semen copper in normal and infertile subjects

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Summary. Concentration of copper in seminal plasma was found to be less than that of normal in cases of oligospermia and azoospermia. It was more in oligoasthenospermia and asthenospermia when compared with that of normal. Chances of initiation of sperm motility by copper is discussed alongwith the inhibitory role it plays.

The role of electrolytes in semen is not well known. The possible role of sodium, potassium, calcium, magnesium[2] and zinc[3] in semen has been discussed earlier. Association of copper deficiency in diet and infertility is well established[4-8]. Also, the toxicity of copper on uterus[9] and spermatozoa[10] is reported. Keeping all these reports of experimental studies in mind, we estimated the copper in seminal plasma in normal and in infertile cases to know if this element has a part to play in the viability of spermatozoa.

Material and methods. 1 sample each from 55 subjects was collected, between 09.00 and 11.00 h, onto clean and dry glass bottles after an abstinence of at least 5 days. The age of the subjects varied from 23 to 35 years. Samples were classified into 5 groups according to its sperm count and motility, as shown in table 1.